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TITLE: Interleukin-1beta expression and phospholipase A2  
activation after intestinal **ischemia/  
reperfusion injury**.  
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AB The experiments were carried out to explore the interactions between IL-1  
beta gene expression, protein level and phospholipase A2 (PLA2) inhibition  
after intestinal **ischemia/reperfusion injury**  
. Using a rat intestinal **ischemia/reperfusion  
injury** model, after collecting the serum, lung lavage, abdomen  
cavity lavage and important organ tissue samples from control, injury and  
PLA2 inhibitor treated groups, IL-1 beta level was measured by  
radioimmunoassay, and the mRNA expression of IL-1 beta and **type  
II PLA2** was determined by RT-PCR. After 6 h of injury,  
the IL-1 beta level in serum was significantly higher than that in the  
control group; an increase in IL-1 beta was also observed in abdomen  
cavity lavage 1 or 3 h after injury. IL-1 beta was significantly increased  
in liver tissue after injury, but was not changed obviously in the lung,  
kidney and intestinal tissues. IL-1 beta in the lung lavage was  
significantly higher than that of control group. The mRNA expression of  
IL-1 beta in lung tissue was increased after injury, but **type  
II PLA2** mRNA expression was decreased. There were  
different changes in IL-1 beta level and gene expression after treatment  
with PLA2 inhibitor chloroquine, cyclo-oxidase inhibitor indomethacin, or  
PAF receptor antagonist SR27417 respectively after injury. All these  
results indicate that after intestinal **ischemia/  
reperfusion injury**, the IL-1 beta level and mRNA gene  
expression are significantly increased, however, the relationship among  
IL-1 beta, PLA2 activation and its metabolite release remains to be  
further elucidated.

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ACCESSION NUMBER: 2001:330655 BIOSIS  
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TITLE: Attenuation of ischemia and reperfusion injury of canine  
livers by inhibition of type II phospholipase A2 with  
LY329722.  
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SUMMARY LANGUAGE: English  
AB Background. Membrane phospholipid breakdown, caused by ischemia and  
reperfusion (I/R) of the liver, releases free fatty acids including

arachidonic acids and lysophospholipids, which serve as precursors of various inflammatory lipid derivatives. Phospholipase A2 (PLA2) is a key enzyme that initiates this reaction. In this study, we tested our hypothesis that a **type II PLA2** inhibitor, LY329722, could attenuate hepatic I/R injury caused by a 2-hr total hepatic vascular exclusion (THVE) in dogs. Methods. Eighteen beagle dogs, subjected to a 2-hr THVE, were divided into three groups. Group 1 (n=6) was untreated and served as a control group. LY329722 was administered to animals in group 2 (n=6) intravenously (0.2 mg/kg) for 60 min before ischemia, and to animals in group 3 (n=6) for 60 min starting 15 min before reperfusion (0.2 mg/kg). Animal survival, systemic and splanchnic hemodynamics, hepatic tissue blood flow, liver functions, energy metabolism, hepatic venous thromboxane B2 and endothelin-1 levels, phospholipid levels and tumor necrosis factor- $\alpha$  mRNA expression in liver tissue, and histopathologic findings were evaluated. Results. Two-week animal survival was 33% (two of six) in group 1, and 100% (six of six) in groups 2 and 3. LY329722 improved systemic and splanchnic hemodynamics, hepatic tissue blood flow, and energy metabolism, reduced liver enzyme, thromboxane B2, and endothelin-1 release, prevented hepatic phospholipid degradation and tumor necrosis factor- $\alpha$  mRNA expression, and lessened histopathologic damage and the number of neutrophil infiltrating into the liver tissue. Conclusion. The present study demonstrated that a **type II PLA2** inhibitor, LY329722, attenuated hepatic I/R injury caused by a 2-hr THVE model in dogs.

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